



STIC Search Report

Biotech-Chem Library

STIC Database Tracking Number: 121920

To: Sarvamangala Devi
Location: REM 3C18
Art Unit: 1645
Monday, May 17, 2004

Case Serial Number: 10/030529

From: Beverly Shears
Location: Remsen Bldg.
RM 1A54
Phone: 571-272-2528

beverly.shears@uspto.gov

Search Notes

Shears, Beverly

From: Devi, Sarvamangala
Sent: Thursday, May 13, 2004 7:43 AM
To: Shears, Beverly
Subject: 10/030,529

Beverly:

Would please perform a sequence and an interference search for SEQ ID NO: 1 and SEQ ID NO: 2 and fragments thereof in application SN 10/030,529? Please also run the DNA sequence against the amino acid sequence.

Please include an inventors' name search: Christopher Elkins

Thanx.

S. DEVI, Ph.D.
AU 1645
Rems - 3C18

10/030529

13may04 08:12:11 User219783 Session D2016.2
SYSTEM:OS - DIALOG OneSearch
File 65:Inside Conferences 1993-2004/May W2
(c) 2004 BLDSC all rts. reserv.
File 440:Current Contents Search(R) 1990-2004/May 13
(c) 2004 Inst for Sci Info
File 348:EUROPEAN PATENTS 1978-2004/May W01
(c) 2004 European Patent Office
File 357:Derwent Biotech Res. 1982-2004/May W2
(c) 2004 Thomson Derwent & ISI
File 113:European R&D Database 1997
(c)1997 Reed-Elsevier(UK)Ltd All rts reserv
*File 113: This file is closed (no updates)

Set	Items	Description	Author
Set	Items	Description	
S1	8	AU=(ELKINS, C? OR ELKINS C?) AND (DSRA OR DSR(S)DUCREYI OR DUCREYI (5N) RESISTANCE)	
S2	5	RD (unique items)	

>>>No matching display code(s) found in file(s): 65, 113

2/3,AB/1 (Item 1 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)
(c) 2004 Inst for Sci Info. All rts. reserv.

14916377 Document Delivery Available: 000178675100030 References: 44
TITLE: The Haemophilus **ducreyi** serum **resistance** antigen
DsrA confers attachment to human keratinocytes
AUTHOR(S): Cole LE; Kawula TH (REPRINT); Toffer KL; **Elkins C**
AUTHOR(S) E-MAIL: kawula@med.unc.edu
CORPORATE SOURCE: Univ N Carolina, Dept Microbiol & Immunol, Campus Box 7290/Chapel Hill//NC/27599 (REPRINT); Univ N Carolina, Dept Microbiol & Immunol, /Chapel Hill//NC/27599; Univ N Carolina, Dept Med, /Chapel Hill//NC/27599
PUBLICATION TYPE: JOURNAL
PUBLICATION: INFECTION AND IMMUNITY, 2002, V70, N11 (NOV), P6158-6165
GENUINE ARTICLE#: 605JQ
PUBLISHER: AMER SOC MICROBIOLOGY, 1752 N ST NW, WASHINGTON, DC 20036-2904 USA
ISSN: 0019-9567
LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: Haemophilus **ducreyi** is the etiologic agent of the sexually transmitted genital ulcer disease chancroid. H. **ducreyi** serum **resistance** protein A (**DsrA**) is a member of a family of multifunctional outer membrane proteins that are involved in resistance to killing by human serum complement. The members of this family include YadA of Yersinia species, the UspA proteins of Moraxella catarrhalis, and the Eib proteins of Escherichia coli. The role of YadA, UspA1, and UspA2H as eukaryotic cell adhesins and the function of UspA2 as a vitronectin binder led to our investigation of the cell adhesion and vitronectin binding properties of **DsrA**. We found that **DsrA** was a keratinocyte-specific adhesin as it was necessary and sufficient for attachment to HaCaT cells, a keratinocyte cell line, but was not required for attachment to HS27 cells, a fibroblast cell line. We also found that

10/030529

DsrA was specifically responsible for the ability of *H. ducreyi* to bind vitronectin. We then theorized that **DsrA** might use vitronectin as a bridge to bind to human cells, but this hypothesis proved to be untrue as eliminating HaCaT cell binding of vitronectin with a monoclonal antibody specific to integrin alpha(v)beta(5) did not affect the attachment of *H. ducreyi* to HaCaT cells. Finally, we wanted to examine the importance of keratinocyte adhesion in chancroid pathogenesis so we tested the wild-type and **dsrA** mutant strains of *H. ducreyi* in our swine models of chancroid pathogenesis. The **dsrA** mutant was less virulent than the wild type in both the normal and immune cell-depleted swine models of chancroid infection.

2/3,AB/2 (Item 2 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)
(c) 2004 Inst for Sci Info. All rts. reserv.

12441699 References: 41

TITLE: **DsrA**-deficient mutant of *Haemophilus ducreyi* is impaired in its ability to infect human volunteers

AUTHOR(S): Bong CTH; Throm RE; Fortney KR; Katz BP; Hood AF; Elkins C ; Spinola SM (REPRINT)

AUTHOR(S) E-MAIL: sspinola@iupui.edu

CORPORATE SOURCE: Indiana Univ, Dept Med, 435 Emerson Hall,545 Barnhill Dr/Indianapolis//IN/46202 (REPRINT); Indiana Univ, Dept Med, /Indianapolis//IN/46202; Indiana Univ, Dept Microbiol & Immunol, /Indianapolis//IN/46202; Indiana Univ, Dept Pathol & Lab Med, /Indianapolis//IN/46202; Indiana Univ, Dept Dermatol, /Indianapolis//IN/46202; Univ N Carolina, Dept Med, /Chapel Hill//NC/27599; Univ N Carolina, Dept Microbiol & Immunol, /Chapel Hill//NC/27599

PUBLICATION TYPE: JOURNAL

PUBLICATION: INFECTION AND IMMUNITY, 2001, V69, N3 (MAR), P1488-1491

GENUINE ARTICLE#: 404GZ

PUBLISHER: AMER SOC MICROBIOLOGY, 1752 N ST NW, WASHINGTON, DC 20036-2904 USA

ISSN: 0019-9567

LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: *Haemophilus ducreyi* produces an outer membrane protein called **DsrA**, which is required for serum resistance. An isogenic **dsrA** mutant, FX517, was constructed previously in *H. ducreyi* 35000. Compared to its parent, FX517 cannot survive in normal human serum. When complemented in trans with a plasmid containing **dsrA**, FX517 is converted to a serum-resistant phenotype (C, Elkins, K, J, Morrow, Jr., and B, Olsen, Infect. Immun, 68:1608-1619, 2000). To test whether **dsrA** was transcribed in vivo, we successfully amplified transcripts in five biopsies obtained from four experimentally infected human subjects. To test whether **DsrA** was required for virulence, six volunteers were experimentally infected, with 35000 and FX517 and observed for papule and pustule formation. Each subject was inoculated with two doses (70 to 80 CFU) of live 35000 and 1 dose of heat-killed bacteria on one arm and with three doses (ranging from 35 to 800 CFU) of live FX517 on the other arm. Papules developed at similar rates at sites inoculated with the mutant or parent. However, mutant papule surface areas were significantly smaller than parent papules. The pustule formation rate was 58% (95% confidence interval [CI]

10/030529

of 28 to 85%) at 12 parent sites, and 0% (95% CI of 0 to 15%) at 18 mutant sites ($P = 0.0004$). Although biosafety regulations precluded our testing the complemented mutant in humans, these results suggest that expression of **DsrA** facilitates the ability of *H. ducreyi* to progress to the pustular stage of disease.

? log y

13may04 08:19:52 User219783 Session D2016.3